

# Epidural Anaesthesia for LSCS in Hypertrophic Cardiomyopathy: A Better Choice.

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## ABSTRACT

Hypertrophic cardiomyopathy (HCM) is a complex cardiovascular disorder with autosomal dominant inheritance, having an incidence of 0.1-0.5% in pregnant females. Anesthetic management of female with HCM posted for caesarean section is a challenge, as even minor alteration in hemodynamic status during the perioperative period may endanger the life of the patient. Here we report successful management of one patient with HCM for LSCS using epidural anaesthesia.

**Keywords:** Hypertrophic cardiomyopathy (HCM), Left ventricular outflow tract (LVOT), LSCS, Epidural Anaesthesia.

## INTRODUCTION

Hypertrophic cardiomyopathy (HCM) with or without left ventricular outflow tract (LVOT) obstruction during pregnancy is a challenge for anaesthesia management. It is characterized by asymmetric hypertrophy of the interventricular septum (IVS) causing intermittent obstruction of the left ventricular outflow tract (LVOT). The condition is worsened by decrease in the preload or afterload or by sympathetic stimulation leading to an increase in myocardial contractility. This is commonly encountered during anaesthesia and surgery. In addition, the higher incidence of ischemic heart disease in patients with HCM may further increase the risk.<sup>[1]</sup> Sudden unexpected death, presumably caused by acute LVOT obstruction or a fatal cardiac dysrhythmia, is possible in an asymptomatic patient.<sup>[2]</sup> The overall incidence of HCM is 1:500 (0.2%); whereas incidence is 0.1 to 0.5% in pregnancy.<sup>[3]</sup> Here we are presenting a diagnosed case of HCM posted for emergency LSCS done successfully under epidural anaesthesia

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## CASE REPORT

A 23 years old second gravida female with average height and weight 48 kg, was admitted to the hospital at 32 weeks of gestation with grade II breathlessness. She had no history of chest pain, syncopal or presyncopal attacks. Her family history was not supportive of any inheritance. She had previous history of abortion at 2 months of gestation, however no details were available. Her past history was not significant. Her exercise

tolerance was normal before pregnancy. Her pulse rate was 100/min, BP was 130/80 mmHg. She had bilateral pedal edema and the JVP was raised at the time of admission. Her breath sounds were clear. She had an ejection systolic murmur in aortic area. Her ECG was suggestive of left axis deviation with T inversion in avL, V1-V6. 2D Echo revealed asymmetric interventricular septal hypertrophy (HCM) without significant gradient with mild aortic regurgitation. The biventricular systolic function was normal. Interventricular septum thickness was 20 mm and ejection fraction was 80%. On her cardiologist referral she was advised Tablet Lasix 40 mg BD which she discontinued after three days on the disappearance of symptoms. The further progress of her pregnancy was uneventful. She came to the hospital in labor after four weeks (36 weeks) and was posted for emergency LSCS in view of cephalo-pelvic disproportion. Considering her previous reports, she was investigated for routine haemogram, KFT and LFT which were unremarkable. She had no breathlessness this time. On evaluation her pulse rate was 80/min; BP was 124/78 mmhg, the JVP was not raised; Chest was clear and ejection systolic murmur was heard in aortic area. ECG findings were same as before. High risk consent was obtained. Anti-emesis prophylaxis was given with IV 4 mg Ondansetron and IV 50 mg Ranitidine. Under antibiotic coverage she was preloaded with 400 ml Ringer's lactate (RL) solution. Multipara monitor was applied consisting of Pulse Ox, NIBP, ECG. Urine output was monitored by urinary catheter. CVP line was not inserted due to lack of time.

Under all aseptic precautions, 18 G epidural catheter was inserted in left lateral position in L3-L4 space and test dose was given with 2 ml of 2% lignocaine. The patient was made supine and a wedge was placed below her right buttock. Slow and titrated epidural top-up doses (total 12 ml) of 0.5% Bupivacaine were given. The desired sensory level of T6 was achieved after 15 min. Oxygen was

administered by facemask. A healthy, 2.6 kg baby, with an APGAR score of 9/10, was delivered. IV 15 units of Pitocin was started slowly. She was haemodynamically stable throughout the procedure. The patient was sedated with IV 1 mg of Midazolam and 40 mcg of Fentanyl. A total of 1250 ml of IV fluids were infused intra-operatively; urine output was 400 ml. Postoperative analgesia was achieved by Fentanyl patch. Epidural catheter was removed. Patient was shifted to post anesthesia care unit for observation and monitoring, and was shifted to the ward the next day. Patient was discharged uneventfully on the 12th postoperative day.

## **DISCUSSION**

Hypertrophic cardiomyopathy (HCM) is a heterogenous disorder produced by mutation in multiple gene coding for sarcomeric protein.<sup>[4]</sup> It is autosomal dominant and almost 150 mutations have been identified so far. It can present during any phase of life from infancy to old age. The family trait is seen in almost 70% of cases but sporadic cases are not uncommon.

The main abnormality in hypertrophic cardiomyopathy is asymmetric hypertrophy of inter-ventricular septum (IVS) in the area of left ventricular outflow tract, which may or may not be associated with hypertension or aortic stenosis. The hypertrophy is more common in upper IVS, below aortic valve leading to LVOT obstruction. This is aggravated by anterior motion of the septal leaflet of mitral valve known as SAM.<sup>[5]</sup> The degree of severity of Hypertrophic cardiomyopathy is decided by the thickness of inter-ventricular septum. The normal thickness is 12 mm or less. 13-15 mm is denoted as mild hypertrophy while 30-60 mm is considered as severe. Although some people who carry a mutant HCM gene, may have normal wall thickness.<sup>[4]</sup> In our patient the wall thickness was 20 mm with good ejection fraction.

Pathophysiologically anterior asymmetrical hypertrophy is associated with anterior motion of mitral valve causing dynamic obstruction to left ventricular outflow tract. The impaired isovolumetric relaxation time and filling of stiff and hypertrophic left ventricle causes diastolic dysfunction seen in almost 80% of patients and it represents an important determinant of clinical course. The narrowing of coronaries due to hypertrophy may cause ischemic heart disease presenting with dyspnoea and chest pain. The cellular disarray results into disorganized ventricular architecture leading to arrhythmogenesis. Atrial fibrillation is the most common arrhythmia and it is poorly tolerated with an associated high risk of thromboembolism. The myocardial disarray and fibrosis are the histological hallmarks of the disease. All these factors can complicate the haemodynamic balance of the patient. The presence of obstruction

portends poor prognosis as compared to non-obstructive type.<sup>[6]</sup> Family history of sudden death, left ventricular wall thickness >30 mm, unexplained syncope and ventricular tachyarrhythmia with thromboembolism are the major risk factors for sudden cardiac death.

HCM patients usually tolerate pregnancy well.<sup>[7]</sup> In pregnant patients the increase in volume load causes enlargement of the ventricular cavity. Along with which the raised cardiac output increases the left ventricular outflow tract gradient causing distension of left atrium resulting into atrial fibrillation. While during delivery, auto-transfusion by contracting uterus, further increases cardiac output. Heart rate can also be increased due to pain, stress or blood loss. All these physiological changes lead to an increase in LVOT gradient and shorten the diastolic filling period, therefore increasing the risk of pulmonary oedema. Though pregnancy is contraindicated in patients having significantly impaired systolic function (left ventricular ejection fraction <40%) or in severe outflow tract obstruction, surgical correction of obstruction can fulfil the desire of pregnancy.<sup>[8]</sup>

Diagnosis of HCM is done primarily on 2D Echo. Salient features to note on echo are measures of diastolic function, systolic function, localization and severity of hypertrophy, outflow tract gradients at rest and with provocation, degree of mitral regurgitation and left atrial dimensions. Although cardiac magnetic resonance imaging has an additional role, particularly if the hypertrophy is isolated to the LV apex and it can also diagnose the presence and severity of myocardial fibrosis. In our case, the patient had mild aortic regurgitation but the gradient was insignificant and left ventricular ejection fraction was 80%. The degree of LV outflow tract obstruction is assessed by continuous-wave Doppler echocardiography, by virtue of the characteristic midsystolic peaking waveform. A recent positron-emission tomography study showed that the degree of microvascular dysfunction in patients with HCM is an independent predictor of heart failure and death and may precede clinical deterioration by many years.<sup>[9]</sup>

ECG shows ST-T wave abnormality due to left ventricular hypertrophy. Deep narrow q wave (dagger like) in V5 -6, I, avL is characteristic of asymmetric septal hypertrophy. The size of Q wave differentiates ischemia with HCM. >40 ms of Q wave will go in favour of ischemic heart disease, whereas <40 ms is seen in HCM. P-mitral is seen in left atrial hypertrophy. In our patient, there was left axis deviation and T inversion in avL, V1-V6 suggestive of left ventricular asymmetric hypertrophy. Monitoring in the perioperative period is of prime importance in HCM. Anticipation of dynamic LV obstruction, malignant arrhythmias, myocardial ischemia, maintaining intravascular fluid volume and systemic vascular resistance are the

mainstay. Intraoperative transoesophageal echocardiography may also be useful for examining dynamic changes in cardiac performance and the response to inotropes and fluid loading.

Anesthetic goals should be to minimize sympathetic stimulation, expand intravascular volume, and prevent increases in left ventricular afterload. LSCS can be performed under general anesthesia as volatile agents decrease myocardial contractility which is advantageous but risk of reversible congestive heart failure and the tendency of volatile agents to produce junctional rhythms mandates caution.<sup>[10]</sup> An elective caesarean delivery may be performed safely with epidural anesthesia.<sup>[11]</sup> Regional anaesthesia has the advantage of reducing afterload which can improve cardiac output. However, hypotension must be prevented to avoid myocardial hypoperfusion. When an epidural or spinal anaesthesia is administered judiciously, the benefits of pain reduction and reduced sympathetic stimulation may outweigh the disadvantages of these regional techniques.<sup>[12-14]</sup> Oxytocin must be administered carefully because of its vasodilating properties and compensatory tachycardia. Pulmonary edema has been observed in parturients with HCM after delivery, emphasizing the delicate fluid management.

## CONCLUSION

Hypertrophic cardiomyopathy in pregnancy was once thought to be malignant disease with high risk of sudden death; now proved to be benign condition demanding proper perioperative care. It may be worth emphasizing that successful anesthetic management of a patient with HCM under epidural anaesthesia requires thorough understanding of the hemodynamic changes, proper intraoperative vigilance, avoiding factors that may increase LVOT obstruction with proper medication and intravascular fluid therapy. Family screening and genetic counseling can definitely improve the outcome in pregnant patients.

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**How to cite this article:** Bengali R, Patil T, Pawar R. Epidural Anaesthesia for LSCS in Hypertrophic Cardiomyopathy: A Better Choice. *Ann. Int. Med. Den. Res*. 2016;2(1):46-8.

**Source of Support:** Nil, **Conflict of Interest:** None declared